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Case report

Pseudoaneurysm originating from left ventricle aneurysm: An autopsy case and review of literature

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ABSTRACT

Rupture of the free wall of the left ventricle is a catastrophic complication of acute myocardial infarction. Rarely, free wall rupture is contained by overlying adherent pericardium, producing a pseudoaneurysm of the left ventricle. In this report, a case of a left ventricular pseudoaneurysm due to a previous myocardial infarction is described. A 55-year-old woman had a severe chest pain 11 months prior to death. No cardiac investigation was performed. Three days prior to death, she suffered from fatigue and weakness, and had a witnessed sudden cardiac death. At autopsy, a 8.5 × 10 × 8 cm pseudoaneurysm of the left ventricle was found. There was severe coronary artery atherosclerosis. There were extensive adhesions between pericardium and pseudoaneurysm wall. The cause of death was attributed to heart failure and resulting arrhythmia. The case illustrates the rare event of left ventricular pseudoaneurysm first diagnosed at forensic autopsy.

1. Introduction

Perforation or rupture of the left ventricular free wall is a dramatic and often fatal complication that typically occurs three to five days after the onset of acute myocardial infarction.[1] Rarely, adhesions of epicardium and pericardium, and a parietal thrombus at the region of infarct may lead to the formation of pseudoaneurysm (PSA) (also known as false aneurysm) in which the walls are formed by pericardium and the organized thrombotic material.[1–3] Left ventricular PSA are prone to rupture, however, there are occasional reports of prolonged survival of a patient with an unruptured left ventricular PSA.[4–6] Patients who had PSA after myocardial infarction tended to present with recurrent anginal chest pain, congestive heart failure and rarely with arrhytmia, syncope and systemic embolism.[7] In this paper, a case of left ventricle PSA determined at autopsy is evaluated together with the data in the literature.

2. Case report

A 55-year old woman of low socio-economic class, poor access to health care, and a 60+ pack-year smoking history, had an episode of severe chest pain 11 months prior to death. However, she didn't apply to any medical unit with this complaint. Instead, she had ten-day rest at home. For the last two months she had complaints about feeling of tightness in her chest, getting tired easily and edema on her legs. She was in bed for the last 3 days, suffering from fatigue and weakness. After the dinner she got worse and died when her spouse was with her. As she had been at odds with her husband, her death was considered suspicious. In the autopsy, there observed reddish–purple color changes in the apex region of the pericardia. There were extensive adhesions between pericardia and inferolateral part of left ventricle (Fig. 1). The heart weighed 280 g. The right coronary artery, left anterior descending branch of left coronary artery, and the left circumflex artery were hardened by severe atherosclerosis and calcification. Old infarct region causing a scar tissue containing all of the myocardial layers of the apex of the left ventricle and the septum and an aneurysm with a diameter of 8.5 × 10 × 8 cm was determined. There was organized thrombus in the aneurysm (Fig. 2). There were 200 cc serous liquid in both chest cavities. The histopathologic examination revealed

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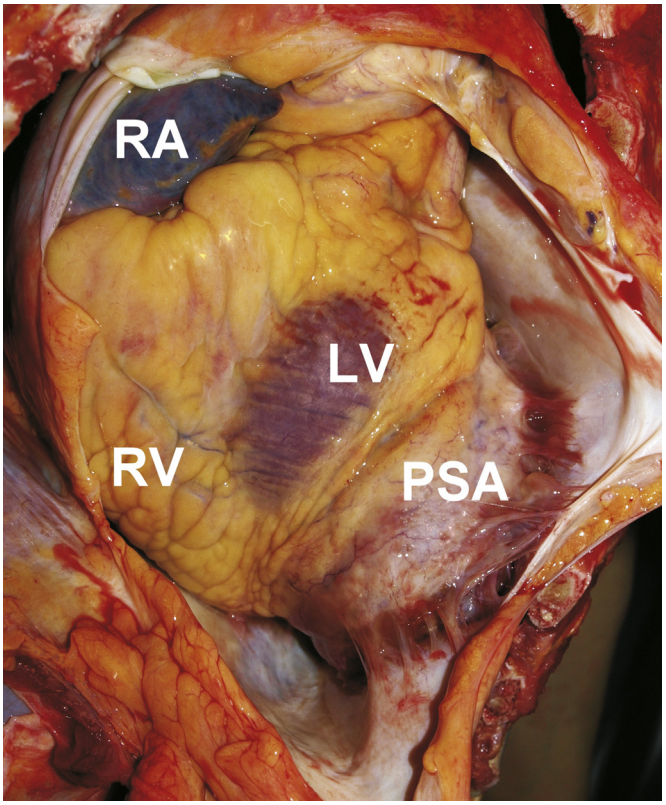

Fig. 1. Extensive adhesions between pericardia and inferolateral part of left ventricle. PSA: Pseudoaneurysm, LV: Left ventricle, RV: Right ventricle, RA: Right atrium.

that there was an additional PSA originating from the inferior part of the aneurysm as this region was containing only pericardial and fibrous elements in its wall, myocardial tissue couldn't be observed. It was concluded that the cause of her death was as a result of heart failure occurred due to the PSA developed after myocardial infarction.

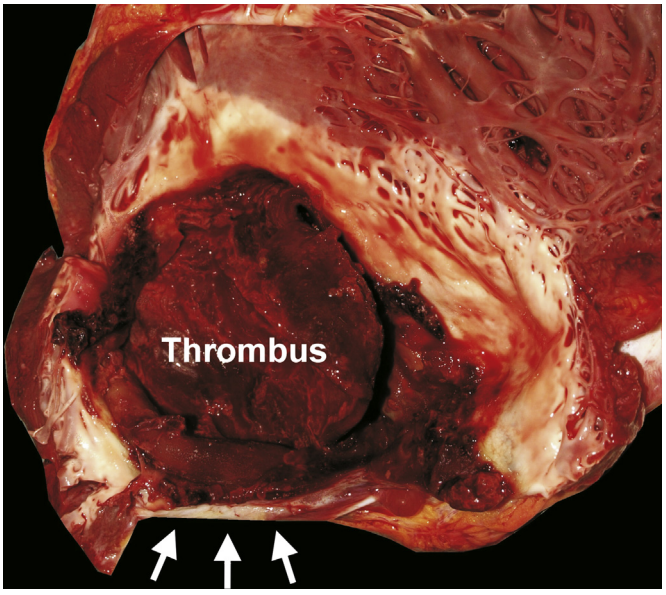

Fig. 2. Organized thrombus in the aneurysm, arrows are indicating the thin wall of pseudoaneurysm.

3. Discussion

Rupture of the free wall of the left ventricle is a catastrophic complication of myocardial infarction, occurring in approximately 4% of patients with infarcts and in about 23% of those experiencing fatal infarcts. PSA of the left ventricle, which in fact is a contained rupture, is rare, because in most instances ventricular free wall rupture is usually associated with sudden cardiac death because of haemopericardium and subsequent cardiac tamponade.[8–10] A ventricular PSA most often occurs in transmural infarctions (55%). Other etiologies include surgery (33%), trauma (7%), or infection (5%).[3,11,12]

In 1967, Roberts and Morrow[13] reported the case of a patient with and reviewed the literature on postinfarction ventricular PSA. They noted six prior reports, the earliest by Corvisart in 1797.[1,14]

Left ventricular PSA usually presents with symptoms, but more than 10% of patients can be asymptomatic.[3,11] Diagnosis is complicated because the most frequently reported symptoms are heart failure, chest pain and dyspnea, all of which are common in patients with coronary artery disease. In addition, patients also have nonspecific complaints such as cough, altered mental status, and dizziness, which rarely elicit a concern for a left ventricular PSA.[11,15]

Pseudoaneurysms are often detected incidentally by echocardiography or other imaging modalities. Because of their propensity to rupture, it has been recommended that they be repaired surgically. They may also give rise to congestive heart failure since the cavity is noncontractile, or to embolic events, because of the stagnant flow of blood leading to thrombosis.[16,17]

In some patients the diagnosis of PSA is made many years after myocardial infarction.[2,17] There are occasional reports of prolonged survival of a patient with an unruptured left ventricular PSA.[4] In 1963, Hurst et al.[18] reported a patient who survived six years with PSA after acute posterolateral myocardial infarction. Congestive heart failure is the most common presentation, followed by angina, ventricular arrhythmias, and embolization.[2,19] The case presented in this study died before the PSA diagnosis was set up, after 11 months of myocardial infarction.

Pseudoaneurysms have been reported to originate usually at the posterior basal and rarely at the apical segment of the left ventricle as seen in our case.[20,21]

Histopathological examination of excised ventricular wall helps to confirm the diagnosis of this disease.[22,23] Unlike true aneurysms, which are regions of dilated ventricle encompassing all the layers (i.e. the endocardium, myocardium and epicardium), ventricular pseudoaneurysms affect only the epicardium and are often contained by the serosal layer of the pericardium.[1,24] Histopathologic examination was performed in our case and PSA diagnosis was confirmed.

It was found that, two patients died of congestive heart failure,[16,25] two died of documented arrhythmias[26] on review of the primary reports. All of these deaths may have been attributable to PSA. Past complaints and postmortem findings of our case support heart failure.

Thrombus is frequently found in the left ventricular PSA.[27] Embolization of thrombotic material, induced by stagnant patterns of blood flow, has also been reported with large pseudoaneurysms (>3 cm in diameter).[11] In one study, 13% of the patients with a left ventricular PSA had systemic embolism as the clinical presentation.[28] There was thrombus in the left ventricular PSA in our case, also.

Unlike true aneurysms, which have a resistant fibrotic wall, pseudoaneurysms initially consist of loose tissues and have an excessively high propensity for secondary rupture. Most investigators have supported surgery as the appropriate treatment

for left ventricular PSA since untreated pseudoaneurysms have an approximately 30%–45% risk of rupture.[4,11,16]

4. Conclusion

This case report illustrates the rare event of previously undocumented left ventricular pseudoaneurysm first diagnosed at forensic autopsy. It is important to distinguish between pseudoaneurysm and true aneurysm especially by determining the presence of myocardium surrounding the cavity. The presence of myocardium surrounding the cavity suggests true aneurysm and myocardial discontinuity suggests pseudoaneurysm.[1]

Although PSA is a complication that can cause sudden death due to rupture and heart failure, it has been thought that the life duration of a patient may be extended by preventing any sudden death resulted from myocardial rupture at an early stage with a kind of repair mechanism by PSA. If the diagnosis is done and the treatment is given after the development of PSA, the life duration of a patient will be longer. Nevertheless, as in our case, if the diagnosis is not done and if any treatment is not given, it may become a fatal complication.

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Conflict of interest
None.

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